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Case Report

Overlapping Hyperemesis Gravidarum, Gestational Thyrotoxicosis, and Transaminitis in Early Pregnancy: A Case Report

Urwah Noor[1], Muhammad Shumas[2], Amal Shahzad Khan[3], Rizwan Zafar[4], Javeria Rizwan[2,*]

1-FMH College of Medicine & Dentistry, Lahore, Pakistan
2-Akhtar Saeed Medical & Dental College, Lahore, Pakistan
3-Foundation University Medical College, Islamabad, Pakistan
4-Department of Medicine, Akhtar Saeed Medical & Dental College Lahore, Pakistan

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ABSTRACT

Hyperemesis Gravidarum (HG), characterized by excessive emesis, fluid depletion, electrolyte imbalance, and weight loss, is a condition that affects approximately 0.3-2% of pregnancies. In this case report, we highlight the importance of careful investigation, evaluation, and supportive management of a primigravida woman diagnosed with HG along with concurrent development of gestational transient thyrotoxicosis (GTT) and HG-associated transaminitis at ten weeks of gestation. HG typically starts in the first trimester, during which GTT is commonly observed. A 23-year-old primigravida in her first trimester of gestation presented with persistent vomiting, progressive fatigue, and an inability to maintain oral intake. Upon investigation, her liver enzymes were elevated, and thyroid function tests (TFTs) revealed a classic picture of transient hyperthyroidism, with negative thyroid receptor antibodies (TRAb)/thyroid-stimulating immunoglobulin (TSI). Upon ruling out other causes, she was diagnosed with HG-associated transaminitis complicated by GTT. She was managed supportively with IV fluids, antiemetics, and thiamine. Due to the self-limiting nature of GTT, anti-thyroid drugs were not initiated. She was then discharged on Day 7 in a vitally stable condition. At the eight-week follow-up after discharge, symptoms and laboratory abnormalities improved. Recognizing the diagnostic complexity of overlapping HG, GTT, and HG-associated transaminitis is crucial for appropriate conservative management.

1. Introduction

Nausea and vomiting are commonly experienced during early pregnancy; however, a small percentage of pregnant women develop hyperemesis gravidarum (HG), a much more severe condition characterized by restless vomiting, fluid depletion, ketonuria, and weight loss. This condition affects around 0.3-2% pregnancies and often requires hospitalization for supportive treatment [1].

In some instances, HG is accompanied by transient thyroid gland overactivity, known as gestational transient thyrotoxicosis (GTT). Since human chorionic gonadotropin (hCG) mimics thyroid stimulating hormone (TSH), elevated hCG levels can cause a GTT, which is self-limiting once hCG levels decrease [2].

Furthermore, up to 50% of women with HG may have mild to moderate liver enzyme elevations, reflecting hepatic stress due to malnutrition or volume contraction. Generally, these changes resolve quickly once hydration is adequate and vomiting ceases [3]. Individually, all these conditions are well-documented in the literature, but their overlap poses significant challenges for diagnosis and management. For instance, a false interpretation of GTT as Graves' disease could lead to the unnecessary use of antithyroid drugs, while overlooking hepatic implications could delay appropriate resuscitative actions. We report a case of a primigravid woman at 10 weeks of gestation, highlighting the importance of careful evaluation and supportive management to ensure maternal and fetal well-being.

2. Case Presentation

A 23-year-old primigravida woman in her first trimester (10th week) of pregnancy presented with an 8-day history of persistent nausea and vomiting (8-10 episodes per day), progressive fatigue, weight loss of 3 kg, and an inability to retain oral intake. The history of fever, abdominal pain, change in bowel movements, and oliguria was negative. The patient denies alcohol use, herbal supplements, hepatotoxic medications, and excessive acetaminophen intake. There was no prior history of liver, thyroid, or bone disease. The physical examination was unremarkable except for a dry tongue, which indicated dehydration. The hematological findings **(Table 1)** were within the normal range with hemoglobin 12.8 g/dL (12 – 16), total leukocyte count 11.0 $\times 10^3/\mu L$ (4.3 – 11.0), neutrophilic predominance (58%), lymphocytes 19%, platelets 317 $\times 10^3/\mu L$ (150 – 450 $\times 10^3/\mu L$), and the international normalized ratio (INR) **(Table 2)** was 1.0 (0.8-1.1). Liver function tests **(Table 3)** showed ALT 404 U/L (7 – 56), AST 111 U/L (8 – 40), alkaline phosphatase 135 U/L (44 – 147), total bilirubin 1.7 mg/dL (0.3 – 1.2 mg/dL), and Albumin

* Corresponding author: Javeria Rizwan, Akhtar Saeed Medical & Dental College, Lahore, Pakistan. Email: urwahnoor2904@gmail.com

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Table 1: Complete Blood Count (CBC) at Presentation

Parameter	Value	Reference Range
Hemoglobin (g/dL)	12.8	12–16
Total Leukocyte Count (×10³/μL)	11.0	4.3–11.0
Neutrophils (%)	58	40–70
Lymphocytes (%)	19 ↓	20–45
Platelets (×10³/μL)	317	150–450

CBC, complete blood count; ↓, below reference range.

Table 2: Coagulation Profile at Presentation

Parameter	Value	Reference Range
INR	1.0	0.8-1.1

INR, international normalized ratio.

Table 3: Liver Function Tests

Parameter	First Report	Second Report	Reference Range
Total Bilirubin (mg/dL)	1.7 ↑	0.5	0.3–1.2
ALT/SGPT (U/L)	404 ↑	54	7–56
AST/SGOT (U/L)	111 ↑	43	8–40
ALP (U/L)	135	129	44–147
Albumin	3.8	3.9	3.5–5.0

ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase; ↑, above reference range.

3.8 g/dL (3.5 – 5.0 g/dL). Viral hepatitis serologies (HBsAg, anti-HCV, anti-HEV IgM) were negative. Autoimmune and cholestatic markers were not performed because there was no clinical or imaging evidence of autoimmune liver disease or cholestasis. Urinalysis (dipstick) showed ketonuria (small/1+; <20 mg/dL), consistent with mild starvation ketosis secondary to reduced oral intake and persistent vomiting. Thyroid function tests (TFTs) were measured using standard automated chemiluminescence immunoassay analyzers in a clinical laboratory setting. They showed suppressed TSH (0.02 mIU/L) with elevated total T3 (5.05 nmol/L) and total T4 (440.8 nmol/L), exceeding trimester-specific reference ranges **(Table 4)**. Repeat immunoassay **(Table 5)** confirmed elevated free T4 at 51.7 pmol/L (10-22 pmol/L) and free T3 at 11.87 pmol/L (3.8-6.6 pmol/L) with suppressed TSH (0.03 mIU/L). Thyroid Autoantibody Assays **(Table 6)** were performed, which revealed negative TRAb/TSI, ruling out Graves' disease with TRAb at 0.8 IU/L (<1.75 IU/L) and TSI at 89% (<140%). Electrolytes showed Sodium (Na^+) 136 mmol/L (135–145 mmol/L), Potassium (K^+) 3.3 mmol/L (3.5–5.0 mmol/L), Magnesium (Mg^{2+}) 1.9 mg/dL (1.7–2.2 mg/dL), Chloride (Cl^-) 92 mmol/L (95–105 mmol/L), Bicarbonate (HCO_3^-) 30 mmol/L (22–28 mmol/L) and venous pH was 7.46 (7.35–7.45) monitored daily during hospitalization **(Table 7)**. Quantitative serum Beta-hCG was 121,046 IU/L (44,186–170,409 IU/L), appropriate for a 10-week gestation **(Table 8)**.

Table 4: Thyroid Function Tests – Baseline

Parameter	Value	Reference Range (1st trimester-specific)
Total T3 (nmol/L)	5.05 ↑	1.8-4.3
Total T4 (nmol/L)	440.8 ↑	77-231
TSH (mIU/L)	0.02 ↓	0.1-2.5
Free T3 (pmol/L)	11.87 ↑	3.8-6.6
Free T4 (pmol/L)	51.7 ↑	10-22

T3, triiodothyronine; T4, thyroxine; TSH, thyroid stimulating hormone; ↑, above reference range; ↓, below reference range.

Table 5: Thyroid Function Tests – Follow-up

Parameter	Value	Reference Range (1st trimester-specific)
Total T3 (nmol/L)	3.9	1.8-4.3
Total T4 (nmol/L)	175	77-231
TSH (mIU/L)	0.15	0.1-2.5

T3, triiodothyronine; T4, thyroxine; TSH, thyroid stimulating hormone.

Pelvic ultrasonography showed a single viable intrauterine pregnancy at 10 weeks of gestation, with normal fetal movement and cardiac activity. The crown – rump length was measured to be 3.4 cm, an appropriate gestational sac size, and normal placental morphology. There was no evidence of molar pregnancy or gestational trophoblastic disease. Abdominal ultrasound showed a normal-sized liver with preserved echotexture and no focal lesions. The portal vein and biliary tree were normal. The gallbladder, spleen, pancreas, and kidneys were unremarkable. Thyroid ultrasound showed a normal-sized thyroid gland with preserved echotexture. There was no evidence of any nodules, cysts, or increased vascularity.

The patient was admitted and received supportive and symptomatic treatment. She received 2 L of intravenous Ringer's Lactate upon admission for rehydration, with daily monitoring of electrolytes, including potassium, sodium, and magnesium. She was given Thiamine 100 mg IV once daily for three days before the introduction of dextrose-containing fluids to reduce the risk of Wernicke's encephalopathy. Mild hypokalemia was managed with adequate supplementation, with routine monitoring until normalization. For persistent vomiting and nausea, Ondansetron 8mg IV every 8 hours was initiated for three days. Over the first week, her hydration gradually improved, urine output stabilized, and she was shifted to oral fluids, which she was able to tolerate in small, frequent meals. Subsequently, intravenous fluids were tapered off. No antithyroid drugs were administered, as gestational thyrotoxicosis was suspected and thyroid function was expected to normalize spontaneously. Venous thromboembolism prophylaxis was not initiated as it was not indicated, as the patient was not bedridden. The patient was discharged on Day 7 in a stable condition with a net weight gain of 1.5 kg and a negative repeat urine ketone test.

At the 8th-week post-discharge follow-up, liver function improved, and laboratory findings supported recovery. Thyroid function also improved, and the patient continued her pregnancy without further

Table 6: Thyroid Autoantibody Assays

Parameter	Value	Reference Range
TRAb (IU/L)	0.8	<1.75
TSI (%)	89	<140

TRAb, TSH receptor antibody; TSI, thyroid stimulating immunoglobulin.

Table 7: Electrolytes, Acid-Base Status, and Urine Ketones at Presentation

Parameter	Value	Reference Range
Sodium (mmol/L)	136	135-145
Potassium (mmol/L)	3.3 ↓	3.5-5.0
Magnesium (mg/dL)	1.9	1.7-2.2
Chloride (mmol/L)	92 ↓	95-105
Bicarbonate (mmol/L)	30 ↑	22-28
pH	7.46 ↑	7.35-7.45
Urine Ketones	Small / 1+ (<20 mg/dL)	Negative

↑, above reference range; ↓, below reference range.

complications. The patient had hyperemesis gravidarum with deranged liver function tests in association with transient gestational thyrotoxicosis. All clinical and biochemical abnormalities resolved with supportive treatment, without the need for antithyroid drugs. Ethical approval for this case report was obtained from the Institutional Review Board of Akhtar Saeed Medical & Dental College, Lahore, Pakistan. The case report is fully de-identified, and no patient identifiers or identifiable images are included.

3. Discussion

This case highlights the rare overlapping presentation of gestational thyrotoxicosis, hyperemesis gravidarum, and HG-associated transaminitis in a primigravida woman in her first trimester of pregnancy. Although each of these conditions has been described individually, their coexistence brings insight into the challenges clinicians face when distinguishing between self-limiting diseases like these and conditions that require targeted therapy.

Gestational thyrotoxicosis is a transient, self-limiting form of thyrotoxicosis that occurs during early pregnancy. This rare disorder occurs in approximately 0.2% of pregnancies [4]. It is characterized by hyperthyroxinemia with an elevated free thyroxine (T4) and suppressed serum thyroid-stimulating hormone (TSH) [5]. The underlying mechanism of this pathology is primarily due to thyroid-stimulating hormone (TSH) and human chorionic gonadotropin (hCG), both of which have a homologous structure [6]. Increased hCG levels during early pregnancy can cause hyperthyroidism secondary to TSH-receptor activation [7].

Differentiating between GTT and autoimmune diseases like Graves' disease (GD) is essential, as management strategies differ substantially, particularly in the absence of clinical features suggestive of GD. Our patient profile was typical of this condition. She presented with elevated free T3, T4, and suppressed TSH, which indicated hyperthyroidism. Thyroid antibody tests were performed, and GD was excluded due to a negative result. Our findings aligned with

Table 8: Serum Beta-hCG at Presentation

Parameter	Value	GA-specific Reference Range
Beta-hCG (IU/L)	121,046	44,186 - 170,409 (10 weeks GA)

GA, gestational age; Beta-hCG, beta human chorionic gonadotropin.

GTT, rather than autoimmune hyperthyroidism, which would have required a different therapy.

Hyperemesis Gravidarum (HG) is a poorly understood complication that arises during early pregnancy and is characterized by persistent and severe nausea and vomiting [8]. It occurs in around 0.3-2% of pregnant women [9]. It is often incorrectly referred to as "morning sickness", but its clinical significance often surpasses this trivialization [10].

Another striking feature of this case was the degree of elevation in liver enzymes. Studies have reported that pregnant women with HG often have significant increases in liver enzymes compared to pregnant women without HG [11]. Our patient presented a similar clinical picture, with elevated ALT, AST, and moderate hyperbilirubinemia. However, serological studies were unremarkable, and the most plausible explanation was HG-associated transaminitis.

Limitations include the lack of bilirubin fractionation and additional cholestatic markers (e.g., direct bilirubin, GGT, or bile acids), as well as the absence of autoimmune liver serology. Nevertheless, negative viral serologies, normal INR and albumin, unremarkable imaging, and clinical improvement with supportive HG management supported HG-associated transaminitis as the most likely diagnosis.

The management of HG complicated by GTT and HG-associated transaminitis warrants a careful balance of interventions and diagnostic vigilance, and in our patient, hospitalization helped in achieving that. We were able to administer intravenous fluids, correct the electrolyte balance, and administer thiamine to prevent Wernicke's encephalopathy, a complication of prolonged HG [12]. Treatment with antiemetic medications is standard in this disease according to recent recommendations [5], so Ondansetron was administered after a risk-benefit assessment [13].

Considering that gestational thyrotoxicosis is usually self-limiting and resolves by itself as hCG levels fall, its management is typically supportive with clinical and biochemical monitoring. We tailored our treatment accordingly [14]. We opted out of antithyroid medication in accordance with recent recommendations since serum T4 returns to normal by 14 – 18 weeks of gestation, and the use of these drugs during early pregnancy can have teratogenic effects [14]. Our patient's improvement with supportive care alone underscores the importance of distinguishing between these diseases.

This case raises broader reflections on patient care, tackling the overlap of gastrointestinal, endocrine, and hepatic symptoms, known to cause much diagnostic uncertainty, and the patient may suffer from physical and psychological distress throughout the process. Hence, timely diagnosis and reassurance are necessary for effective treatment and to prevent further complications. This interdisciplinary collaboration among obstetrics, endocrinology, and hepatology enabled a holistic approach, ensuring both maternal and fetal well-being.

In summary, this case illustrates the triple presentation of gestational thyrotoxicosis, hyperemesis gravidarum, and HG-associated

transaminitis in early pregnancy. It highlights the significance of ruling out alternative etiologies, timely supportive management to control symptoms, and awareness about the natural history and progression of these conditions. As our understanding of these diseases expands, we should focus on developing more targeted therapies that might ease the burden of these conditions for patients, enabling better, more effective patient care.

4. Conclusion

This case highlights the severe overlap of HG, GTT, and HG-associated transaminitis in early pregnancy, which, if not carefully differentiated, could potentially complicate diagnosis and management. Our patient's recovery through conservative management without antithyroid drugs emphasizes the need to recognize the self-limiting nature of GTT readily. A high index of suspicion, prompt diagnosis, interdisciplinary collaboration, and patient-centered care were crucial for ensuring both maternal and fetal well-being in our case. In conclusion, this study aims to raise awareness of the growing need for increased vigilance in managing such atypical cases in early pregnancy.

Conflicts of Interest

The authors declare no conflicts of interest.

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Informed Consent

Written informed consent for publication was obtained from the patient.

Large Language Model

Generative artificial intelligence (ChatGPT, OpenAI) was used to assist with language refinement and grammar. All authors reviewed and verified the content and take full responsibility for the manuscript.

Authors Contribution

UN and MS drafted the manuscript and compiled case details. ASK contributed to the literature review and drafting. RZ provided clinical supervision and revision. JR coordinated the journal submission. All authors approved the final manuscript.

Data Availability

The data supporting the findings of this case report are contained within the article. No additional de-identified patient data are available for sharing due to privacy and confidentiality considerations.

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